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Chronic Wasting Disease and the Science in support of the Ban on Baiting and Feeding Deer.

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Summary

Reliable science provides support for a ban of baiting and feeding of white-tailed deer to reduce disease risks for Chronic Wasting Disease (CWD). Peer-reviewed research papers published in reputable scientific journals indicate the following:

- **CWD is transmitted laterally (live diseased deer infect other deer)**
- **Deer can get CWD by ingesting something contaminated with the disease prion**
- **CWD prions may be shed in feces and saliva**
- **Disease course and symptoms indicate high potential for transmission where deer are concentrated**
- **Evidence from captive situations indicates that deer can get CWD from highly contaminated environments.**
- **Baiting and Feeding causes *unnatural* concentration of deer**
- **Reduction of contact through a ban on baiting and feeding is likely very important to eradicating or containing a CWD outbreak.**
- **Baiting and feeding continues to put Wisconsin's deer herd at risk to other serious diseases**

In addition, experts in CWD, wildlife disease and deer nutrition support bans on baiting and feeding as part of a comprehensive strategy to prevent and/or manage CWD.

Under a baiting and feeding ban, disease outbreaks are more likely to be smaller in scale and more apt to be contained or eliminated. With the long CWD incubation period and other factors that make discovery of a new outbreak difficult, an outbreak that is already widespread when detected because of baiting and feeding may not be able to be contained or eliminated.

This document provides details and explicit links to the supporting science.

Chronic Wasting Disease and the Science behind the Ban on Baiting and Feeding Deer.

Introduction

A healthy, robust white-tailed deer herd is important to Wisconsin hunters, wildlife watchers and businesses. A year ago, three deer in the Mt. Horeb area were discovered to have chronic wasting disease (CWD) - a transmissable and always-fatal disease of the nervous system.

CWD threatens Wisconsin's deer herd. It's a complicated problem with no easy solutions. The Department of Natural Resources has proposed strong actions to eradicate the disease in Wisconsin, including a ban on baiting and feeding of deer - a practice science tells us elevates the risk of CWD transmission.

A complete discussion of the proposals and their impacts is in a newly published Environmental Impact Statement, also available on the DNR website. In March, the department will hold a series of hearings across the state to gather citizens' opinions on the department's proposed actions.

The following is a partial list of scientific evidence that suggests that baiting and feeding of wild deer elevates the risk of CWD transmission. This list focuses almost entirely on disease risks posed by CWD although other diseases (Bovine Tuberculosis is an example) may pose even greater risks.

Complete literature citations are included at the end of the document for those who want to read the original scientific articles.

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- **CWD is transmitted laterally (live diseased deer infect other deer)**

Researchers who have studied CWD epidemics in both captive and free-ranging deer populations have determined that CWD is both contagious and self-sustaining (meaning that new infections occur fast enough for CWD to persist or increase over time despite the more rapid deaths of the diseased individuals; Miller et al 1998, 2000). Supporting evidence comes from observational data (Williams and Young 1992; Miller et al. 1998, 2000) experimental data, and epidemiological models fit to observed prevalences in free-living deer (Miller et al. 2000, Gross and Miller 2001, M. W. Miller unpublished in Williams et al. 2002). These studies suggest that observed prevalences and rates of spread of CWD in real populations could not occur without lateral transmission. For example, maternal transmission (doe to fawn) if it occurs, is rare and cannot explain most cases where epidemiologic data are available(Miller et al. 1998, 2000). Similarly, indirect lateral transmission (e.g. from a contaminated environment) may require unusually high levels of contamination (see below; Williams et al. 2002). Nonetheless, emerging research from Colorado suggests that indirect lateral transmission from environmental contamination appears to play a role in sustained and recurrent epidemics (Miller 2002).

- **Deer can get CWD by ingesting something contaminated with the disease prion**

Six mule deer fawns were fed a daily dose of 2g (0.07 ounces) of brain tissue from CWD-positive mule deer in a tightly controlled experiment for 5 days. Another three were fed the same doses using brain tissue from CWD-negative mule deer. All deer were held separately in indoor pens that had never before held deer. The fawns were then killed and necropsied at specific intervals 10 to 80 days post-inoculation. At 42 days and later post inoculation, all fawns dosed with CWD-positive tissue tested positive for CWD prions in lymph tissues associated with their digestive tracts (Sigurdson et al. 1999). Other transmissible spongiform encephalopathies (TSEs); Kuru, transmissible mink encephalopathy, bovine spongiform encephalopathy (BSE]) appear to be transmitted through ingestion of prion-infected tissue as well (Weissmann et al. 2002). Due to the human health crisis associated with eating BSE-

infected beef in Europe, many other researchers working with TSEs, including CWD (Sigurdson et al 1999, 2001), have traced the movements of infectious prions of orally-infected animals through the lymph tissue embedded in the intestinal lining, into nervous tissues associated with the digestive tract (Maignien et al 1999, Beekes and McBride 2000, Heggebo et al. 2000, Huang et al. 2002) and eventually to the brain via the nervous system (Sigurdson et al. 2001, Weissmann et al. 2002). Experimental studies using hamsters have shown that prions can infect through minor wounds in the skin (Taylor et al. 1996) and that infection through minor wounds on the tongue was more efficient than infection from ingestion (Bartz et al. 2003). These studies not only demonstrate that an oral route of infection is possible, but are beginning to provide specific details about the pathways involved in the movement of infectious prions into the central nervous system and other organs (Weissmann et al. 2002).

- **CWD prions may be shed in feces and saliva**

Following oral exposure, prions associated with many TSEs (Maignien et al 1999, Huang et al. 2002) including CWD (Sigurdson et al. 1999; Miller and Williams 2002 and Spraker et al. 2002 cited in Williams et al. 2002) both accumulate and replicate in the lymph tissues associated with the gastrointestinal tract – particularly in lymph tissues in contact with the mucosa lining the inside of the intestines (e.g. Peyer's patches, Weismann et al. 2002). In infected deer, CWD prions also accumulate in the pancreas and various other glands of the endocrine system (Sigurdson et al 2001). Experiments with hamsters demonstrated that infectious prions can travel from the brain to the tongue along tongue-associated cranial nerves (Bartz et al. 2003). During digestion, the liver, pancreas, intestinal mucosa, and other glands secrete chemicals needed for digestion (Robbins 1983) and cells lining the inner surface of the intestine continuously die and slough off providing potential physical mechanisms for prion shedding into the intestines (others are likely). This is evidence that infectious prions are likely shed in the feces and saliva (Sigurdson et al. 1999).

- **Disease course and symptoms indicate high potential for transmission where deer are concentrated**

Appearance of CWD symptoms in an infected deer lags initial exposure by a variable time period on the order of roughly 12 months to 24 months or more (E. S. Williams and M. W. Miller unpublished; E. S. Williams, M. W. Miller, and T. J. Kreeger unpublished; cited in Williams et al. 2002). Once clinical symptoms are observed, deer enter a symptomatic phase that may last on average 1 month to 4 months before they invariably die (Williams et al. 2002). Symptoms are initially subtle but eventually include behaviors likely to contaminate a site with bodily fluids (e.g. excess urination, excess salivation including drooling and slobbering, and uncontrollable regurgitation, Williams et al. 2002). Deposition of feces increases with concentration of deer activity. This is both obvious and intuitive and pellet group counts have been used as an index of deer density since the 1940s (Bennet et al. 1940). During winter, northern deer defecate about 22 times a day (Rogers 1987). At least one study (Shaked et al. 2001) has reported detection of an altered form of the infectious prion in the urine of hamsters, cattle, and humans with TSEs. This altered form, while not as virulent, produced subclinical prion infections following experimental inoculation. Shedding of infectious prions is likely progressive during the course of disease from infection to death (Williams et al. 2002). Replication and presence of infectious prions in gut-associated lymph tissue early in the incubation (Sigurdson et al. 1999, Weismann et al. 2002) and epidemiological modeling (M. W. Miller unpublished cited in Williams et al. 2002) suggest that shedding precedes the onset of symptoms in both elk and mule deer.

In this regard, Garner (2001) documented a particularly alarming behavior among deer using frozen feed piles. Deer used the heat from their mouths and nostrils to thaw and dislodge food such that frozen feed piles were dented with burrows made from deer noses. He reported that "Throughout the winter multiple numbers of deer were observed working in and around the same feed piles. I suspect that each deer that feeds this way at a frozen feed pile leaves much of its own saliva and nasal droppings in the field pile at which its working"(Garner 2001, p. 46).

- **Evidence from captive situations indicates that deer can get CWD from highly contaminated environments**.

In addition to direct lateral transmission, researchers suspect that deer can be infected indirectly from contaminated environments. Contaminated pastures "appear to have served as sources in some CWD epidemics although these observations are anecdotal and not yet corroborated by controlled studies" (Miller et al 1998; M. W. Miller unpublished; E. S. Williams, W. E. Cook, and T. J. Kreeger unpublished; cited in Williams et al 2002). The potential for transmission from the environment is a function of the degree of contamination and the resistance of disease prions to chemical breakdown (Williams et al 2001, 2002). Consequently, the highest prevalences recorded for CWD outbreaks have been in captive situations (Williams and Young 1980; Williams et al. 2002) where because of abnormal concentration, indirect and direct transmission likely occur together (Williams et al. 2002). At high concentration, the persistence of the CWD prion in contaminated environments, may be a serious obstacle to disease eradication (Williams et al. 2002).

- **Baiting and Feeding causes *unnatural* concentration of deer**

People use baiting and feeding to concentrate deer for enhanced hunter opportunity or viewing. In northern deer, seasonal concentration in deeryards is a well-known phenomenon (Blouch 1984). However, the potential for close animal-to-animal contact over a feed pile is fundamentally different than the contact yarded deer experience while foraging on natural food. In deeryards, deer eat a variety of woody browse plants and arboreal lichens (Blouch 1984) scattered across a large area. In terms of biomass and nutrition, the best source of browse and lichens may be litter-fall rather than live plant material growing in the understory (Ditchkoff and Servello 1998). Food sources in deeryards (litter and understory plants) are widely distributed over a large area and they are not replaced. Moreover, browse is typically held aloft on the plant stem such that fecal contamination is less likely. Foraging by wintering deer is an optimization process. Energy gains associated with eating need to be balanced against energy costs associated with travel and exposure (Moen 1976). Yarded deer with little or no access to supplemental food maintain relatively large overlapping home ranges (e.g. 110 acres in Minnesota [Nelson and Mech 1981], 480 acres in Michigan [Van Deelen 1995], 318 acres in Quebec [Lesage et al. 2000]) suggesting that foraging widely on a diffuse food source is normal. Garner (2001) monitored 160 radio-collared deer for 2 fall/winter periods in northern Michigan and documented their behavior over feeding sites using both telemetry and direct observations. He demonstrated that, relative to natural forage, supplemental feeding caused reduced home range sizes, increased overlap of home ranges in space and time and dramatic concentrations of activity around feeding sites.

- **Reduction of contact through a ban on baiting and feeding is likely very important to eradicating or containing a CWD outbreak**.

Epidemiological models fit to real-world data on CWD outbreaks in mule deer predict that local extinction of infected deer populations is likely (Gross and Miller 2001). The predicted outcomes of these models are highly sensitive to input estimates of the amount of contact between infected and susceptible deer meaning that small reductions in contact rates can dramatically reduce the rate at which prevalence changes during an epidemic (Gross and Miller 2001). Garner (2001) demonstrated that baiting and feeding was associated with deer concentration, extensive face-to-face contacts, and increasing overlap of deer home ranges. White-tailed deer have contacts from social and grooming behaviors apart from contact over baiting and feeding sites (Marchinton and Hirth 1984) but social groups of whitetails tend to be small during most of the year (4 to 6 individuals, Hawkins and Klimstra 1970). Whitetail physiology and behavior are adapted to selective foraging on nutritious plants (Putman 1988). Moreover, social groups tend to exclude one another by using different areas or by using shared areas at different times (Mathews 1989, Porter et al. 1991). Concentration of deer activity over feeding sites increase both direct and indirect contact between groups by increasing home range and core area overlap and by increasing the amount of time that unrelated deer feed in close proximity to each other (Garner 2001).

Eliminating these contacts has added significance because CWD is a uniquely difficult disease to manage and study. There is no treatment and no vaccine. Moreover CWD is difficult to track in a population because of long incubation periods, subtle early clinical sign, a resistant infectious agent, potential for environmental contamination and incomplete understanding of transmission mechanisms. These characteristics make prevention critically important (Williams et al. 2002).

- **Baiting and feeding continues to put Wisconsin's deer herd at risk to other serious diseases**

CWD is not the only infectious disease that threatens Wisconsin's deer herd. One, Bovine Tuberculosis (TB) warrants special attention because the link to baiting and feeding is clear. TB is an infectious bacterial disease that is spread from animal to animal through inhalation of infectious aerosols or ingestion of other infectious body fluids (e.g. saliva). Tuberculosis bacteria can live outside of an animal for as long as 16 weeks on a frozen feed pile (Whipple and Palmer 2000 cited in Garner 2001) and Garner (2001) demonstrated that supplemental food increased close contact among wild deer through a number of mechanisms. Garner (2001) also demonstrated extensive home range overlap between a TB-positive deer and 15 other radio-collared deer in northern Michigan. Recent epidemiological research suggests that baiting and feeding of deer enable the TB outbreak in Michigan to persist and spread and that declines in TB prevalence were associated with a ban on baiting and feeding (O'Brien et al. 2002).

Current attention is focused on the CWD outbreak in southwestern Wisconsin. However, should CWD or other infectious disease show up elsewhere, baiting and feeding are likely to facilitate or enhance an epidemic. Tuberculosis has been confirmed on six captive game farms in Wisconsin and the presence of over 800 captive cervid farms statewide suggests that the disease risks associated with baiting and feeding are not confined to the known CWD-infected area of southern Wisconsin.

- **What do the experts say relative to artificial feeding and CWD and disease transmission?**

A discussion of CWD in a review of the scientific literature on captive deer done for The Wildlife Society (Professional society for wildlife biologists, managers, and researchers; publisher of three premier peer-reviewed scientific journals on wildlife ecology and management)...
"Concentration of deer and elk in captivity or in the wild by artificial feeding may increase the likelihood of transmission between individuals." (DeMarais et al. 2002, p. 6).

In a review of the technical literature on CWD by the top CWD specialists in the world...
"Concentrating deer and elk in captivity or by artificial feed probably increases the likelihood of direct and indirect transmission between individuals. Transmission via contact between susceptible and infectious individuals probably requires more than just transient exposure. Thus, minimal fence-line exposure does not pose excessive risk of transmission; however, prolonged fence-line contact increases the possibility of transmission" (Williams et al. 2002, p.557).

In a peer-reviewed paper on the epidemiology of Bovine TB by the team of veterinarians, epidemiologists, and wildlife researchers working to contain the outbreak in Michigan...
"Previous qualitative examinations of the origins of tested deer already suggested that TB positive animals were more likely to come from the core area. Our new analysis quantifies that risk. The high risk associated with the core coincides with an area of historically prevalent and intensive baiting and supplemental feeding of deer – practices that were likely crucial to the establishment of self-sustaining TB in the deer population" (O'Brein et al. 2002 and citations within).

In oral presentations given to the Texas chapter of the Society of Range Management (Oct. 6 2000) and to the Southeaster Deer Study Group (Feb. 19 2001) by Dr. Robert D. Brown, Professor and Head of the Department of Wildlife and Fisheries Sciences at Texas A&M University, Internationally recognized expert on deer and deer nutrition...

"One of the major points of this paper is the concern over transmission of disease. It amazes me that we have not done more studies in Texas on disease transmission at food plots and deer feeders, whether they be for supplementing the deer or for baiting. We know that in 1994 tuberculosis (TB) was first detected in wild deer in Michigan. It is now in a 5-county area, and has spread to carnivores and dairy herds"..."In Wyoming and around Yellowstone Park, brucellosis is wide spread among cattle, elk, and bison, the latter two species being concentrated on feeding grounds in the winter. Likewise, Chronic Wasting Disease (CWD) has now been observed in free-ranging elk and mule deer in several western states. Since CWD is passed animal to animal, concentrations caused by supplemental feeding is believed to increase the spread of the disease" (Brown Unpublished).

In a report issued by a panel of internationally recognized wildlife disease experts who reviewed Colorado's CWD management program...
"Regulations preventing...feeding and baiting of cervids should be continued" (Peterson et al. 2002).

In a comprehensive review of the ecological and human social effects of artificial feeding and baiting of wildlife prepared by the Canadian Cooperative Wildlife Health Centre, Department of Veterinary Pathology, University of Saskatchewan...
"Significant ecological effects of providing food to wildlife have been documented through observation and experimentation at the individual, population, and community levels. The increased potential for disease transmission and outbreak is perhaps of greatest and immediate concern; recent outbreaks of bovine tuberculosis and chronic wasting disease in Canada and the United States giving credence to this point. Nevertheless, even if disease is prevented, other significant ecological concerns exist" (Dunkley and Cattet 2003, p. 22).

Review
To insure that this document accurately reflects the scientific knowledge of prion disease, CWD, and deer biology, this document was reviewed by the following specialists (position and expertise follows each name):

- Judd Aiken Ph.D. (Professor of animal health and biomedical sciences, UW-Madison; *prion diseases*)
- Valerius Geist Ph.D (Professor Emeritus, Department of Environmental Science, University of Calgary; *ecology behavior and management of deer*)
- Julia Langenberg DVM (Wildlife Veterinarian, Wisconsin DNR; *CWD, wildlife diseases*)
- Nohra Mateus-Pinilla DVM, Ph.D. (Research Epidemiologist, Illinois Natural History Survey, University of Illinois; *wildlife diseases, epidemiology*)
- Nancy Mathews Ph.D. (Assoc. Professor of wildlife ecology, UW-Madison; *deer ecology and behavior*)
- Keith McCaffery M.S. (Deer specialist, Wisconsin DNR, retired; *deer ecology and management*)
- Robert Rolley Ph.D. (Population Ecologist, Wisconsin DNR; *population dynamics, deer management*)

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